LETTERS TO THE EDITORS

Lack of precision of duplex scan velocity measurements can falsely elevate rates of progression of carotid stenosis

To the Editors:

Serial duplex scanning has become almost exclusively used to determine rates of restenosis after carotid endarterectomy and the progression of stenosis in unoperated carotid arteries. I have become increasingly concerned that the lack of precision of duplex scans (possibly coupled with intra-observer variability) results in an overestimation of the rates of progression of carotid stenosis when analyzed with actuarial techniques, such as the Kaplan-Meier method. The precision, not accuracy, of an instrument is the variability of repeated measurements of the same subject by the same observer. When sharp cutoff velocity values or velocity ratios are used to separate ranges of degree of carotid stenosis, such as <50% from ≥50%, forward survival analysis of sequential measurements can result in an erroneous rate of progression as the result of duplex scan imprecision. For example, in a recent publication, the calculated annualized rate of progression of carotid stenosis was 9.3% per year for 7 years.[1] The authors report the reproducibility of duplex scan results for the same level of stenosis to be 92% for 48 carotid arteries studied within 1 month. This means that four of the 48 had a different finding and perhaps half or 4% had a higher or progression value. How much of the 9.3% per year was caused by lack of precision? A similar recent analysis indicated a 5.3% per year carotid artery stenosis progression rate.[2] Unfortunately, few studies report the precision of the duplex scanning equipment used or the intra-observer variability. I am unaware of either recognition or of attempts to prevent or correct for imprecision when applying this type data to a survival analysis.

Precision for my office duplex scanner. For 81 unoperated internal carotid arteries measured within 8 weeks by the same technician with an Ultra Mark 9 scanner (ATL, Bothell, Wash), I found a mean absolute difference between two peak systolic velocity (PSV) measurements of 17.5 cm/s and a mean absolute percent difference of 15.1% (range, 45 to 281 cm/s; mean, 123 cm/s). This is similar to a recently reported intra-observer difference of 13.6% for PSV in 20 carotid arteries.[3] Linear regression of my data for the standard deviation (SD) of the difference in two PSV measurements versus PSV gives a SD of 9.0 ± 0.12 PSV cm/s. For example, this regression equation predicts that a PSV of 130 cm/s has a SD of 24.6 cm/s and a 95% confidence interval of 81 to 179 cm/s for the next measurement. If 150 cm/s is used as a cutoff between <50% stenosis and ≥50% stenosis, there is an 18% chance that the next measurement will be 150 cm/s or more.

Theoretic prediction of future peak systolic velocity values when the measured current value is less than 150 cm/s. I used this precision data to theoretically predict what would happen to 600 carotid arteries with actual initial PSV measurements less than 150 cm/s (101 ± 24.5 cm/s, mean ± 1 SD). All initial values were measured by the same technologist with the office machine. Subsequent measurements are predicted by $V_i = V_o + SD \times ND$ cm/s, where i = 1 is 6 months, i = 2 is 1 year, i = 3 is 2 years, i = 4 is 3 years etc, V_o is the initial actual measurement, SD is the precision standard deviation, and ND is the normal distribution. These measurements were computed with JMP version 3.1 statistical software (SAS Instituted, Cary, NC). Kaplan-Meier analysis predicts, for a SD of 15 cm/s and a cutoff of 150 cm/sec or more for progression to ≥50% stenosis, a 5.5% theoretical (erroneous) rate of progression at 6 months, a cumulative rate at 1 year of 8.2%, 10.3% at 2 years, 12.7% at 3 years, and 14.7% at 4 years. The 4-year annualized rate is 3.9% per year—all erroneous and caused by imprecision of PSV measurements. If a precision SD of 20 cm/s is used, the 4-year theoretic rate of progression is 5.5% per year. When the previous regression equation for SD is used, the 4-year rate of progression is 4.2% per year. The erroneous rates of progression decrease as more samples of the same initial velocity values are added because of the forward deletion of values that exceed the cutoff in the survival analysis. These examples of erroneous carotid stenosis progression because of lack of duplex scan precision suggest that our current information on both the rates of progression of carotid stenoses and the recurrent stenosis after endarterectomy may be high.

This is a two-way street. An initial measurement is not necessarily a true one and has the same lack of precision as later ones. Initial velocity values above a cutoff value may drop below it on future testing. Forward actuarial analysis does not account for this. However, this variability may come into play in a similar way when analyzing for regression of stenosis.

One way to reduce the precision SD is to take repeated measurements at the same study period, but this is probably not economically feasible. Another approach is to define the precision of the scanner and technologist and correct the Kaplan-Meier curves accordingly. I do not know how to do this, but it is probably not too difficult. The bad news is that early restenosis or progression rates are probably significantly overestimated because only two, three, or four sequential studies have been done. The good news is that the effect of imprecision dampens out the more studies are done (longer times), and late data is probably much more accurate.

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24/41/99464

Regarding "Occluding aortic endoluminal stent graft combined with extra-anatomic axillofemoral bypass as alternative management of abdominal aortic aneurysms for patients at high risk with complex anatomic features: A preliminary report"

To the Editors:

To assess the usefulness of this report by Le Minh and colleagues (J Vasc Surg 1998;28:651-6), more information on the patient selection is necessary. Simply having a short proximal neck (with no data on measurements) is now not a contraindication to endovascular repair of abdominal aortic aneurysms with either an anatomic bifurcated graft or the less anatomic aorta uni-iliac graft, especially with the use of uncovered suprarenal stents that take the attachment zone away from the seal zone.

No information has been given for the diameter of the endoluminal graft/occluder introducer sheath. This was not a percutaneous procedure. If the occluding graft can be positioned just below the renal arteries, this implies that the vessels were not that tortuous. Therefore, why not simply deploy an aorta uni-iliac endovascular graft with a patent lumen. The extra anatomic bypass graft is therefore much shorter (ie, a femorofemoral crossover graft rather than an axillofemoral graft).

Finally, no information is given as to the length of the procedure. Sick patients have a high mortality after long operations.

Given these criticisms I can see no place for this technique in this endovascular era.

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Reply

We have read with interest the comments of Dr Stuart Walker.

Concerning the abdominal aortic aneurysm (AAA) proximal neck in our series, the average length was 12 ± 3 mm (range, 5 to 25 mm), associated with an angulation from 0 degrees to 95 degrees (mean, 62 degrees). Short proximal neck (<20 mm) associated or not to marked angulation has been shown to cause dislodgments of the stent graft followed by proximal leaks.[1,2]

We agree that the use of uncovered suprarenal stents may prevent proximal endoleaks. However, in the study by Wain et al,[1] there were 21% of endoleaks, despite the placing of proximal uncovered stents near or across the renal arteries among patients with AAA treated with endovascular grafts.

As mentioned in our article, the endoluminal graft introducer sheath had a diameter of 18F (internal diameter 18F; length, 600; Balt Co. Montmarency, France) and was introduced on a super stiff guidewire via a femoral artery cut down.

Tortuosity of iliac arteries does not preclude correct placing of the proximal part of the endograft beneath the renal arteries. In our experience, marked tortuosity of calcified iliac arteries may provoke a kinking of the distal part of the endograft, resulting in cigar-shape formation with subsequent stenosis and thrombosis of the device.

The average length of time needed to implant the stent graft was 120 ± 30 minutes. The total length of the procedure (including axillofemoral bypass grafting) was 175 ± 34 minutes.

We emphasize the fact that this procedure is reserved for patients at high risk with short or angulated proximal neck associated or not with tortuous iliac arteries to ensure total exclusion of the AAA.

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Regarding "Percutaneous transluminal angioplasty for the treatment of limb threatening ischemia: Do the results justify an attempt before bypass grafting?"

To the Editors:

We read with interest the article by Parsons et al (J Vasc Surg 1998;28:1066-71). The conclusions drawn by this article, namely that percutaneous transluminal angioplasty (PTA) is not considered to be a primary treatment in patients with infrainguinal disease and limb-threatening ischemia, should, we think, be qualified.

The authors point out that the published reports of infrainguinal PTA in patients with threatened limbs have shown a range of results, with 1-year patency ranging from 12% to 70%. Our paper, which has been quoted in the article[1] actually shows a 24-month symptomatic patency rate of 77% and a hemodynamic patency rate of 78%. The paper also quotes from our article that only 23% of all the critically ischemic patients were treated with percutaneous transluminal angioplasty. This indeed was true of the data that were collected between 1988 and 1991. We would like to refer you to our subsequent publications in which data was collected during a 1-year period (1994).[2] A prospective